Q Talking about immune symptoms, you mentioned arthritis and rheumatism, pale and numb fingers, rash on cheeks that last more than a month, rash from sun other than sunburn, pain on taking deep breaths, protein or albumin in urine and sudden hair loss.

Are all those items I mentioned immune system symptoms, in your opinion?

A Yes.

Q And you characterized them that way in your report?

A That's correct.

Q Which if any of these immune system symptoms would Ms. McNeal have had had she not been exposed to creosote, penta or dioxin?

A I don't believe she had those symptoms absent her exposures to the dioxins, the PAHs and penta, and all of those alter the immune system, giving rise to an increased risk for immune system symptoms. And I don't know of any other factor in her risk picture that would contribute to the immune system problems.

Q Which of the contaminants you've identified caused or contributed to her immune system? Was it penta? Creosote? Dioxin?

A The most well-studied are dioxins. They have been studied extensively for their propensity to disrupt

and cause alterations in the immune system. There is some data with PAHs and penta, but most of the immune system disruption is most likely related to the dioxins.

Q Are you able to be any more specific, that is can you tell me whether if Ms. McNeal had been exposed to just penta and not also PAHs and dioxins, she would have the same immune system symptoms or the same severity of her immune system symptoms?

A If she had just been exposed to PAHs and penta?

Q No, just dioxin and not the others.

A I don't know. I think the important point is that these patients -- this patient in particular was exposed to chemicals and the whole mixture and developed these health problems.

As I said, I don't know of any other risk factors and it doesn't occur in people who smoke, and doesn't occur in -- I mean immune system dysfunction occurs in the general population but in a low prevalence and I don't know of any other risk factors.

Some doctors may say being black there is a slightly increased rate of scleroderma and lupus in blacks. Other studies have not confirmed that. But I have no reason to invoke any causative factor other than her exposure to the environmental pollutants.

Q She doesn't have scleroderma or lupus, does

she?

A No. I said that's an immune system dysfunction, two diagnoses that have been identified.

Q Let's go back for a moment to the neurological symptoms you mentioned on the last page of your report and we'll mark these as exhibits tomorrow.

This is page 13 and you list a series of neurological symptoms. Do you see that?

A Yes, I do.

Q How many of these are based on self reports?

A All symptoms by definition are self reports.

Q For each one of these plaintiffs, you administered a questionnaire?

A That's correct.

Q And you did an examination?

A That's correct.

Q And you reviewed available medical records?

A Yes.

Q Now, some of the symptoms the plaintiffs reported in their interviews with you and on their questionnaires are not confirmed by medical records; is that correct?

A That's correct. There are many symptoms they complain about that are not listed by their doctors in their medical records, correct.

Q Can you tell me which of the neurological symptoms you have listed on page 13 are also reflected in her medical records and which were reported on her visit with you or her questionnaire?

A Well, in February 1998, she was seen by Dr. Harrison who diagnosed anxiety and didn't describe any further symptoms, unfortunately. None of the visits relative to her hypertension have really probed for her symptoms of headache, dizziness, lightheadedness memory disturbance and so forth.

I don't think there is other mention in her medical reports -- and we have a limited number of records and only Dr. Harrison's records and that's all we have.

Q Anxiety is not one of these neurological symptoms that you listed in your summary?

A No, I don't think so. We don't ask that question, per se, but let's look and see something. We do have an anxiety index under the profile of mood states and that reveals she has some slight anxiety and not a tremendous amount.

Q And what was the doctor's name?

A Harrison.

Q So is Dr. Harrison a neurologist or just a general internist?

A Judging by the records -- I didn't list his specialty but I would think that he is -- well, the internist or family doctor.
Q So she reported as a neurological symptom anxiety to Dr. Harrison?
A That's correct.
Q And several years later she reported a longer list of neurological symptoms to you in response to her questionnaire and her examination; correct?
A That's correct.
Q Let's move on to her immune system symptoms. Which of her immune system symptoms have you confirmed by reference to her medical records?
A I don't think any of them have been confirmed by her medical records.
Q Those are all based on her answers to the questionnaire and her visit with you?
A That's correct.
Q The next item you have in your summary is cancer. And the two cancers you mentioned are skin and cervical cancer in situ; is that correct?
A Yes.
Q And both of those were -- correct me if I'm wrong -- identified in medical records at some point; is that right?

A The carcinoma, yes.
Q And looking at her dermatology -- this is part of your summary, page 11 of 13 -- it says right axillary lipoma removed by Dr. Harrison. What's a lipoma?
A A small benign fatty tumor.
Q That's not cancer?
A Right.
Q Then it says papilloma of neck excised. Do you see that?
A Yes.
Q Is that a cancer?
A No. It's a neoplastic growth but not a cancerous one.
Q What type of skin cancer did Ms. McNeal have?
A She didn't. She misstated that and it was squamous cell carcinoma of the cervix that got erroneously listed as a skin cancer.
Q Do you think the reason she listed it erroneously is squamous cell is also a designation of skin cancer?
A Yes.
Q What causes squamous cell cancer of the cervix?
A There is a virus called human papilloma virus which appears to be a co-factor for the development of cervical cancer, and most patients with cervical cancer

have positive HPV in their cervix, but many patients have it without HPV and do not have cervical cancer. So there has to be a co-factor.
Cigarette smoking is identified as a risk factor for cervical cancer, as is exposure to a variety of other chemicals. But the one relevant here is cigarette smoking. PAHs have been found in the cervical mucous and contribute to the risk of cancer in that organ.
Q And Ms. McNeal is or was a smoker?
A She smoked -- what she said was one cigarette per day.
Q Do you think that's right? I wondered if that was a mistake. Was it one pack or one cigarette per day?
A She was a one cigarette a day smoker. Like a lot of black woman that I have interviewed in Mississippi and elsewhere, she is not a big smoker and doesn't sit and puff on them all day long and she has one cigarette basically as a social event when she's with friends or family.
Q And she began when she was 17 years old?
A That's what she said.
Q And continued through the present time?
A She continues to be what I call a very light smoker.

Q And do you believe her smoking contributed to her cervical cancer?
A Yes.
Q Do you believe her cervical cancer is also related to exposure of dioxins, penta or PAHs?
A I believe it's related to those chemicals, as well, yes.
Q So the three factors we have for her cervical cancer are the -- let me ask a foundational question.
Does cervical cancer of this nature ever occur in the absence of the human papilloma virus?
A I don't recall. It's a high percentage. I'm not sure if a hundred percent and more like 90 percent where cervical cancers occur with HPV positivity.
Q And the HPV is the reason women get Pap smears on a regular basis?
A No. They get them to detect cancer in the early treatable stage. Patients who do not have HPV get cervical Pap smears done on a regular basis.
Q Why is it called a Pap smear?
A After Dr. Papanicolaou who invented the smear.
Q Fair enough. Just to go back to the question, the three contributing factors we have is the human papilloma virus, the cigarette smoking and exposure to PAHs, dioxins and penta; is that correct?

A Yes.
Q Is there any way to separate out for me the extent to which any of the three causative factors contributed to her getting cervical cancer?
A Her exposure to the PAHs by the history of what I have, one cigarette a day, would be a small contributing factor. Her dose of PAHs, dioxin and penta are far, far greater risk factors than that one cigarette a day.
In fact, in the history of cigarette smoking and cancer, patients that are light smokers usually don't have any significant increase in their cancer risk just from smoking. Assuming she does smoke as light as I'm assuming she did.
With the lung cancer risk, you don't begin to see a significant increase in the lung cancer risk of smokers until they get to about 20 pack years, and there is some increase below 20 pack years, but that's when you see the clear separation.
So social smokers like this, they don't have a big risk for getting cancer and that would extend I think to the cervical cancer. So even though it's probably a contributing factor on top of her very high exposures that took place in the neighborhood from the plant, the major, major cause is her exposure to the plant.

The HPV is a necessary but not sufficient factor and seems to be a co-factor, increases the risk but by itself may or may not have caused the disease to occur.
Q Women who are not exposed to PAH, dioxin and penta do get this type of cervical cancer; is that correct?
A Yes.
Q And do women get this type of cervical cancer who are not exposed to the HPV virus?
A I think I told you my best recollection is it's about 80 or 90 percent of patients with cervical cancer have HPV but not all. And, furthermore, there is a prevalence of patients with HPV who never get cervical cancer, so it's a co-factor but not a hundred percent. In other words, just because you have it, it doesn't mean you'll get the disease.
Q We're talking about HPV as sort of a present or not present type of situation.
Are there different levels of HPV different women have, different doses?
A Different types or different intensity of infection?
Q Yes.
A I don't have information about that issue.

Q Do you know whether intensity of infection or number of virus molecules per picogram of blood is something that influences the rate that women get this type of cervical cancer?
A It probably does. I don't remember the data off the top of my head but it makes sense to me if there is documentation of a heavier viral load, that might increase the risk.
Q Viruses act the same way that some of these environmental toxins do, the heavier the dose and the larger exposure the more likely it is that a person will have a negative effect from the virus?
A Well, the thing that determines the viral load is the patient's immune system. You're right. If the immune system is impaired, allowing the virus to grow, that means the immune system is probably less able to fight off cancer. So the mechanism of which the increased rate of cancer in a higher viral load would most likely reduce the immune system in that patient.
Q Can you tell me, but for exposure to PAH, dioxin or penta if Ms. McNeal would have had cervical cancer?
A I don't how you could say that. What we have is a patient in front of us, and she had -- they never did the HPV virus titer on her so we don't know if she

had that -- the but for was absent the exposure to chemicals from Koppers?
Q Right. Would she have had cervical cancer?
A I think it's unlikely given the fact that the co-factor we've identified is her cigarette smoking. She didn't, as far as I know, have any other exposures that would increase her risk, and we don't know if she has HPV, as far as that goes. I don't know how you can say that she would or would not have cancer absent all the facts here.
Q The next item in your summary is respiratory symptoms, including chronic bronchitis, asthma -- it's on page 13 for your summary of Ms. McNeal.
A Right.
Q Which of these respiratory symptoms do you believe are related to her exposure to PAH, dioxin or penta?
A I believe all are related to her exposure to the chemicals here.
Q Were they all caused by the exposure to the chemicals or contributed to by the exposure to chemicals?
A The only risk factor we have here for the respiratory symptoms is her cigarette smoking, which we indicated is quite minimal. You know, I think we should probably look at the other doctors' histories about the

cigarette issue because I think it would help us.
Q Do you have that handy?
A I don't know if I can find it quickly. Dr. Sawyer did a history and let's see --
Q Is this Dr. Sawyer's report?
A No, Dr. Wilson. Dr. Sawyer didn't have any comment about the cigarettes.
Q Did Dr. Wolfson examine Ms. McNeal?
A Yes. The blood pressure was 174 over 84. I don't see a comment about cigarettes and -- there it is. Ms. McNeal smoked for 32 years, starting at age 18, and she continues to smoke one pack per day and tried to stop smoking with use of nicotine patches without success. Her husband is a 2-pack a day smoker since age 16 and stopped at age 55 in November 2004.
So when she wrote one, she meant one pack a day and my comment about her cigarettes would have to be modified.
Q Sure, absolutely.
MR. LUNDY: This is her deposition summary?
THE WITNESS: Yes. There is a lot of smoking history in her deposition.
BY MR. HOPP:
Q This summary is compiled by whom?
A I think Mr. Lundy's firm, one of their

employees.
MR. HOPP: Now that you've shown it, I'd like to make a request for a copy.
MR. LUNDY: He hadn't read it and he'll not rely on it. He can rely on the other doctors' stuff.
BY MR. HOPP:
Q She's obviously also got a significant secondhand smoke exposure?
A From her husband, yes.
Q That's going to contribute to the development of cervical cancer; correct?
A Yes, that's correct.
Q And it's also going to contribute to the development of respiratory symptoms?
A Definitely.
Q Is there any way you can separate out her 26 or more year pack history --
A Pack year history.
Q Her 32 pack year history from her creosote, penta or dioxin exposure with respect to her respiratory symptoms?
A I would say they're both contributing significantly. I don't think we can put a percentage on it or even -- I think both contributed significantly to it. That's based on what we've found in our earlier

study in Columbus, and what we see in this group, as well, that respiratory problems are a big problem associated with living under these conditions, but we also know that patients developed bronchitis and shortness of breath and other respiratory symptoms from cigarette smoking, as well.
Q It's not really possible for you to say that she'd have these identical respiratory symptoms had she never lived in the Carver Circle area; correct?
A I wouldn't want to opine on what would have happened under those theoretical circumstances.
Q I think you answered this, but would it surprise you if someone presented with a 32 pack year history and also had the respiratory symptoms listed for Ms. McNeal on page 13 of 13 on your summary?
A Would it surprise me to have all these symptoms if a 32 pack year smoker?
Q Yes.
A Well, let's get more detail. According to my history which, by the way, is in my handwritten notes --
Q Which you've produced?
A Yes. She started with her asthma ten years before and was using 3 inhalers and two pills.
Q Ten years before what?
A Ten years before this examination and was

experiencing significant shortness of breath in recent years. That history is more characteristic of an asthmatic patient.
It's true that probably the cigarettes would contribute to that but clearly her health condition changed about ten years prior to the examination, significantly, requiring her to go on all those medications, and I think, you know, that's a significant factor and is very common with patients that we've seen living closer to these wood treatment plants and develop a picture like this.
Usually a cigarette smoker, they gradually get worse and don't develop classical asthma-type symptoms and usually don't present in this way and it's usually a gradual buildup, and hers is more like something damaged her lungs in a more acute basis and what it was, I'm not sure. I wouldn't put her in the category of a typical smoker in terms of her history.
Q Are you prepared to render an opinion as to what her clinical picture would look like for respiratory symptoms had she not lived in the Carver Circle area?
A Well, I think I can say without fear of contradiction that she'd have less respiratory problems if she didn't live there.
Q How much less?

A I don't know a number.
Q And you don't know how to take symptoms either on or off the list based on living at Carver Circle; correct?
A In terms of respiratory symptoms?
Q Yes.
A Most patients with cigarette smoking don't complain of eye irritation and reduced sense of smell. They can have some of the other symptoms but those are not typical of a cigarette smoker. So I think they're fairly typical of our patients in Columbus and here, so I believe that those things are probably related to the exposure.
Q Is it your testimony that cigarette smokers don't have a reduced sense of smell?
A They don't complain about a reduced sense of smell in my experience.
Q Let's move on to the next series of symptoms, the dermal symptoms, page 13 of 13 of your summary. She complains of skin redness, dryness, itching and rash on cheeks lasting for more than a month. Which if any of those symptoms are related to her exposure to PAHs, dioxins and penta?
A I think I already indicated I don't see any reason why she would have any skin problems. Cigarette

smoking doesn't do it and exposure to these chemicals does, and I don't see any other contributing factor to her skin complaints.
Q Don't some people just have red, dry skin?
A Not most black people.
Q So it's your opinion that the skin redness, dryness and itching and rash were all caused by exposure to PAHs, dioxin and penta?
A There are patients and even blacks that have an unknown cause for their skin complaints, and they do occur, but in this case I don't have any known other causes, and we do know these chemicals have an effect on the skin, particularly the creosote and naphthalene and other particulates that dry and defat the skin.
Q The studies of creosote and napththalene and drying, those all looked at the application of creosote to the skin; is that right?
A Yes.
Q But that's a transitory effect?
A No. The studies done even by the creosote industry show these skin changes are permanent.
Q In humans or animals?
A Humans.
Q What studies?
A Early studies that were done and they called

them creosote warts and called the photosensitivity a creosote-induced problem and the dry, itchy skin was definitely a permanent effect.
Q Those were studies of workers?
A That's correct. And we found the same thing in our studies of the residents living near the creosote plant in Columbus, Mississippi.
Q Dry and itchy skin?
A Yes, a very common complaint.
Q Ms. McNeal didn't have a creosote wart, did she?
A No.
Q And did you examine her skin for defatting?
A I did not notice any defatting of her skin and this was a subjective complaint and you can't measure defatting in any way I know of or observe it. It's drying of the skin that's reported.
Q As you sit here now, do you remember whether her skin looked scaly and dry?
A No, I don't recall.
Q And the next sentence on page 13 and 13 says that Ms. McNeal's above-noted health problems have been caused by exposure to chemicals from Koppers. Now, the summary of problems we see on page 13 of 13 in your report are all of the problems you

attribute to Ms. McNeal -- all of the problem for Ms. McNeal that you attribute to her exposure to PAHs, penta and dioxin?
A Yes.
Q We covered everything with Ms. McNeal?
A Yes.
Q What sorts of problems would you expect of someone who had severe hypertension and smoked a pack of cigarettes a day to have? Do the two factors working together cause any sorts of health effects?
MR. LUNDY: Other than what he testified to twice?
BY MR. HOPP:
Q Irrespective of chemical exposure. First, severe high blood pressure and smoking a pack a day who doesn't live near a creosote plant, what sort of problems would you expect that person to have?
A Cardiovascular problems, increased rate of heart disease, stroke, and she's got heart disease and congestive heart failure, which is not surprising. She's got diabetes, which I guess is reasonably well controlled, but she has cardiovascular disease and from smoking I would expect her to have respiratory problems as we've stated. Her glucose when I saw her was 159 and she has mild diabetes.

Q I want to get -- and I'm not sure if there is an answer to this or even studied. Smoking is risky behavior and high blood pressure is a general health problem. Have there been any studies that demonstrate what additional problems a person with high blood pressure would have if she smoked?

A Well, the smoking and the high blood pressure together increase the risk of cardiovascular disease.

Q And you testified about that. Anything else you can think of?

A No, that's the main thing.

Q Let's move on to Kay Hobbs --

A As to Patricia McNeal -- I want to refer to Dr. O'Jile's report. She found that she had problems, cognitive impairment, low intellectual functioning, abnormal neurologic tests, similar to what I found.

You want to go to who --

Q I know you referred to Dr. O'Jile, and I want to explore that thought, have you formed any additional opinions with respect to Patricia McNeal after reviewing Dr. O'Jile's report or do the reports support what you previously --

A They support what I previously stated.

Q Kay Hobbs. I'd like you to generally summarize your opinions with Kay Hobbs.

A In terms of her exposure or health effects from the exposure?

Q In terms of health effects from the exposure.

A Well, she has a picture very similar to Ms. McNeal. She has neurologic, respiratory, autoimmune skin problems and they share a great deal, as many of these patients we will see do.

Q The difference with Kay Hobbs is she's deceased -- or one of the differences?

A We're talking about the health problems she had during life, if you will. Her husband filled out the form for her. She was diabetic also and other significant respiratory problems. She was a non-smoker and she had obviously breast cancer that metastasized to her lung and brain.

The breast cancer was diagnosed in 1998 and we have -- she died of breast cancer at the age of 43, diagnosed at age 41. Her sister died a year before, at the age of 34, Sherrie Barnes. She lived in Carver Circle between 1956 and 2000, basically all of her life, and was married in 1974 at the age of 18, and her father worked at Koppers and then Heathcraft, and I believe those are the factors caused by her exposure.

Q So diabetes and breast cancer?

A Well, I think the diabetes -- I didn't mention

that with Patricia McNeal, and it wasn't a major part of her problem and not a major part of this patient's problems, but we know there is an increased risk in diabetes in patients with dioxin exposures and that's a contributing factor to her health problems. In her case the major thing is the breast cancer.

Q What are the other potential causes of breast cancer? What else causes breast cancer?

A Well, there is now data that shows breast cancer is increased in cigarette smokers.

Q Very recent data?

A The last several years. There's animal data to show the PAHs cause breast cancer, and there is evidence that the birth control pills can increase the risk of breast cancer.

I'm not aware of any other factors, and there has been some attempt to find viruses that might increase the risk, but those have not panned out, and the primary thinking about breast cancer causation is it's due to endocrine disruption, and the breast cancer rate is skyrocketing in the last 40 years in this country, and it's felt to be due to the introduction into our environment of endocrine destructors such as PCBs, dioxins and other chemicals of that type.

I think her exposures are really quite -- you

know, it's classically related to the environment. The most striking thing is the early age that she and her sister both contracted this disease, and it's not like she had aunts and -- let me see if I can find what I'm looking for -- I'm trying to see her family history and I have to refresh my memory.

She did not have any other family members with breast cancer, except her sister. There was her father, I guess, the one who worked in the Koppers plant that developed lung cancer, and there is one other cancer in the family but I'm not sure who it was. Anyway, she doesn't have a family history of breast cancer, except she and her sister so that risk element is not present.

Q The family history has been identified as a risk factor for breast cancer; right?

A That's just what I've been saying. But she didn't have that.

Q I've seen an indication in one of the records, and I'm not sure if this is accurate, that her father had male breast cancer. Does that strike a cord with you?

A I have it listed as lung.

Q We can look for it and talk about it tomorrow.

Does secondhand smoke increase the risk of breast cancer?

A Probably.

Q But for living where she did, would Kay Hobbs have developed breast cancer?
A Well, I think it's unlikely that she would have at this young age developed breast cancer. It's because I think the environment she grew up in and lived in was heavily, heavily polluted with potent carcinogens and that's what led to her very aggressive tumor at a very young age.
I mean, it's quite unusual to have cancer at this young age, and her sister had it even at a younger age and it's unusual to have it at this young age, which in itself is suggestive or indicative of high levels of exposure to carcinogens.
Q I want to point something out and drill it down a bit. I asked you whether Ms. Hobbs would have developed breast cancer had she not lived near the Koppers plant and your response took into consideration her age when she developed breast cancer.
Had she lived --
MR. LUNDY: I'll object to the form of your question, the characterization of his response. I think his response was it's highly unlikely. I think you have to quote him that she would have had cancer but for the environmental factors --
MR. HOPP: That's where I want to go.

Q Had Ms. Hobbs lived longer, do you know whether she would have developed breast cancer irrespective of her exposure to the Koppers plant?
A I have no way of knowing that.
Q Is it your opinion that Ms. Hobbs developed breast cancer at an earlier age than she would have or might have otherwise as a result of her exposure?
MR. LUNDY: Objection; that's ignoring his previous response that he wouldn't know and so I object to the form of the question.
BY MR. HOPP:
Q I'll let the witness clarify his opinion.
A She has no risk factors for developing breast cancer, except for her exposures in the neighborhood. I think the point is there is a risk factor for cancer in general of age. After the age of 60, the rate of cancer of all types increases significantly in the United States. We have no way of saying that she might have developed it when she was 70 or 80 years old.
She has no risk factors for that and it's not an opinion I would hold that she would have, but the point is, in addition to the fact that she developed this tumor at an unusually early age, and her sister even earlier is indicative to my way of thinking of a highly carcinogenic environmental, which all the numbers and

studies we've done have corroborated.
In other words, the finding of a very carcinogenic environment with the testing and then finding patients like Ms. Hobbs living in the midst of this very dangerous environment, it's not surprising that she got cancer at an early age that was very resistant to treatment and died very quickly with metastatic disease.
This is all important for understanding why this environment needs to be remediated immediately.
Q And, again, I apologize if you answered or think you answered it and I didn't understand it.
If Ms. Hobbs had not lived near the Koppers plant, would she have developed breast cancer at all ever?
A I can't say one way or the other. I indicated that it's likely since there is no risk factor for developing breast cancer. If she developed any type of cancer, it's more likely like the general population at a much older age, but I'm not saying she would have. There is no way of saying that.
Q Can you say whether Ms. Hobbs developed breast cancer at an earlier age than she might have otherwise, as a result of her exposure to creosote, penta and dioxins?
A And I'll say it the fourth time, there are no

other risk factors for breast cancer in this patient.
MR. HOPP: Read back the question.
(Record read.)
BY MR. HOPP:
Q I don't understand how your answer is responsive to the question. Can you explain --
A I think the cause of her breast cancer was the exposure. I thought that was clear. I'm trying to be clearer.
Q If her father had breast cancer, would that be enough of a family history for you to believe that her family history might have contributed?
A That's not what the data and the literature shows. It shows increased risk associated with female family members. I don't know of any data that shows that male breast cancer is related in any way to higher risks in the daughters.
But remember, I think this is a father who worked in Koppers plant and he had all the risk factors that she had, and if it was,-- male breast cancer is as rare as hen's teeth, and for him to have that is in itself remarkable and suggests why did he have breast cancer. I would think the answer is because he worked at Koppers and had all the exposures to increase the risk of mammary cancers.

Q Would there also be a genetic predisposition of that?
A I'm not aware of any data to suggest that male breast cancer is associated to genetic factors. Unlike female breast cancer where there is some familial aggravation.
Q You indicated male breast cancer is very rare. Is it studied much?
A No, as a result of it being rare.
Q Let's move on to Derion Griffin.
A I looked at Dr. Wolfson's report on Kay Hobbs and it confirms what I have stated, that there is no other risk factors for the development of breast cancer.
Who is next?
Q Derion Griffin.
A I'm here. He's the little boy that's 9 years old with hydrocephalus.
Q And what are your opinions with respect to the medical conditions or symptoms that Derion Griffin has as a result of exposure to PAH, dioxins and penta?
A Well, he has got, in addition to his hydrocephalous, some of the same symptoms we saw in the other two patients. Neurological symptoms, respiratory symptoms, immune system problems, although limited just to allergies in his case.

He has skin problems and, as I say, the most important is his hydrocephalous which is a developmental problem that I believe is a birth defect that he obtained from exposure to these various agents.
Q Would it be fair to characterize Derion Griffin as mentally retarded?
A Let's see what Dr. O'Jile said about his mental functioning. I think she did test him. She has got a detailed history of his gestation early years. She felt he was moderately to severely impaired and she felt that was superimposed on a background of extremely low intellectual functioning.
He has deficits in adaptive functioning and areas where he did better, and some areas of relative strength, including general fund of information, vocabulary and simple attention, assembling puzzles and receptive vocabulary and drawing of simple figures, gross psycho-motor speed and calculation and with continued supervision throughout his life and given his functioning levels, expectations regarding his education and occupational abilities are low.
In your answer to your question, he appears to be at a level where his ability to function in the labor market and to benefit from education is very, very limited.

Q Do you remember meeting Derion Griffin?
A No.
Q Do you remember whether he was able to respond to your questions in order to fill out the questionnaire that you administered?
A No. He was not able to do that.
Q And his mother did that for him?
A Yes, and also gave the history.
Q And just going back to Kay Hobbs, obviously she is deceased and her husband filled out the questionnaire?
A I have Walter Hobbs, and I believe that's the husband.
Q Are you aware of any literature that is published on the reliability of health status questionnaires when they're filled out by someone other than the patient?
A Well, with children and this young man, even though he's 9, and some 9-year-olds can fill out questionnaires but usually a normal 9-year-old gets a lot of help from his parents and some adult or older child.
Children don't give reasonable questionnaires until probably up to 12, 14, 15 depending on the skill level of the child and understanding the process. I'm not surprised by his inability to fill it out.
Now, in terms of reliability, I'm not aware of

any peer-reviewed study that directly answered that question, and, from my own experience, I think what I found is the mothers tend to be reasonably good observers of their children.
However, in this case, the mother has got a problem with memory herself because she lives in the environment and there are a number of discrepancies between what she said on different occasions about her son. That's I think reflecting her low functioning level in terms of memory and integration of data.
I think if we were to ask the question does this young man have a neurological problem, I don't think anybody would argue he does. Somebody who had hydrocephalous and a shunt placed frequently have problems with cognition and development of the brain.
He has apparent respiratory problems and he's complained about it and has been treated for it. Let me see if I got what I -- the medical records here. It's mainly focused on the neurologic, but he had pulmonary hemorrhages, which is, I guess, another health problem diagnosed in 1995.
When he was born he had to be intubated and he's been on theophylline which is a medicine for respiratory problems like asthma. So there is some objective backing in the records of health problems of

the type the mother describes, albeit somewhat limited.
Q The question, though, went towards the reliability of her responses, and you'll admit, will you not, that when someone's medical history is filtered through another person, there is a risk of some discrepancies in the data that's unreliable?
A I'd agree. But the important point here is when we look at this patient in the context of all the other patients we see, when she says he has cough and brings up phlegm and sometimes he has -- what does she describe here -- sinusitis, it tends to be credible because we can see it in the context of all the other people in the neighborhood who have similar complaints and due to the heavy contaminations in these homes, it's not surprising the people have these complaints.
So taking all the factors into consideration, I think it becomes reasonable to believe -- I mean she doesn't answer all the questions as abnormal, and she has many she claims he never had on the questionnaire, and at least internally and externally putting it all together, it's consistent with the pattern that we're seeing in this group of people.
Q At least in Derion Griffin's case you can look at his medical records and find out what his measured problems are; correct?

A Yes.
Q But the question was broader and deals with Kay Hobbs and Derion Griffin --
A Remember Derion was born prematurely --
Q I will ask you that in a minute and let me finish the question. With respect to Kay Hobbs, her husband filled out the form?
A Yes.
Q And she's been dead for 5 years or so -- for 4 years by the time he filled out the form?
A Yes.
Q And there is a concern that when someone else fills out the form that some data gets lost and some questions may be answered inaccurately?
A I think that's a fair statement.
Q With respect to Derion, he has a list of medical records which you relate back to, it sounds like, primarily to his hydrocephalous?
A Hydrocephalous was sort of his ongoing diagnosis but remember he had a very stormy entrance into life.
Q Let's talk about that. He was -- let me ask a foundational question.
How many weeks is considered term for an infant?

A 40.
Q 40 weeks gestation?
A Yes.
Q How many weeks gestation did Derion Griffin have?
A Well, it's estimated that he had about 22 to 24 weeks of gestation. That's the estimate.
Q What's the general survival rate of an infant born at 22 weeks' gestation?
A Very poor. I don't know the statistics but most of the children born that small don't make it.
Q It's amazing he's alive at all, isn't it?
A It's quite a tribute to the skills of the neonatologist. He was in the hospital for 92 days.
Q Immediately following Derion Griffin's birth, do you know how much time he spent without any medical care?
A After he left the hospital?
Q After he was born. Didn't they think he was dead for a while?
A They said earlier they intubated him right away, and he was not breathing and had to breathe for him. He weight a pound and 11 ounces. He was under two pounds and a very, very small baby.
Q Do you know how long a time lag there was

between the time Derion was actually delivered and when intubated?
A No, I don't have -- his APGAR score was 1 so that means he was not breathing and they had to resuscitate him. Obviously they resuscitated him, and how long he was lying pulse-less or not breathing, I don't know.
Q Do you know how long it was between the time Derion was born and the time he was air-lifted to another hospital for special care?
A It only says he was taken and I don't have minutes or duration of the treatment at the local hospital.
Q Are you in a position to give opinions with respect to the effect that Derion's medical care in the first hours of his life had on his condition later in life?
A It seemed to me that it was a miracle that he survived so his medical care couldn't be too bad. If you're asking if they didn't intubate him quickly enough and allowed anoxic injury to occur due to malpractice, I don't have an opinion about that. There's nothing in the record to suggest that they did anything other than the best that they can do for this baby.
Q I would never suggest otherwise, but do you

know whether there was an injury that Derion sustained -- leaving malpractice out of this completely -- whether there was an injury he sustained because of a lack of oxygen after his birth due to the fact that he was not intubated quickly?

A It says he had apnea of prematurity, and he was not breathing because he was so premature and his breathing mechanisms were not working and I don't know how many minutes it was --

Q You're reading Dr. Wolfson's summary?

A Yes. My summary doesn't have any information about that either. I mean, I just don't know what to tell you.

Q That's fair. But you're not taking that issue into account with respect to your opinions? That is you're not factoring in the notion that he might have been caused an injury by the failure of the medical staff to intubate him early in life?

A There is no question that he had anoxia and that he was not breathing. How many minutes without oxygen is not clear from the record. Very premature babies like this do, in fact, have anoxia by definition and getting the oxygen into the blood stream so they survive when separated from the mother is very important. If there is a prolonged period of anoxia that can cause

permanent brain damage. But I have no reason to know that happened or didn't happen.

I think there was also the question of hemorrhages at the time of birth and definitely a pulmonary hemorrhage, but I think there was also a question about bleeding into the brain, which is common in these children.

One of the children we saw in Columbus, Mississippi was very premature like this, and he had pulmonary hemorrhage that resulted in severe mental changes. Very premature babies like this generally have a lot of problems and one of them is anoxia and one of the mechanisms by which they get problems.

Q I understand your answer but I need to go back to my question.

Is it accurate to say that you're not taking into account for the purpose of your opinions any knowledge or assumption with regard to how long Derion went without intubation after he was born?

A As I said, I don't have any information how many minutes it was.

Q And you assume he received the appropriate medical treatment for a baby his size and age?

A I believe so, yes. I believe they tried their best to do what they could for this baby.

Q Why was Derion Griffin born prematurely?

A Well, there is a powerful risk factor which we know about and that's his exposure to PAHs. Some very elegant studies have now been done by Dr. Perera on PAH levels in the cord blood and small gestational age could occur and apply here and also prematurely. That's the mechanism that cigarette smoking mother have smaller babies is because of the PAHs in cigarettes.

They've now shown that PAHs in the environment -- Dr. Perera's studies of babies in Poland, which has a lot of PAH contamination, showed a very strong correlation of the PAH content of the mother and the prematurity of the baby, and I think in this case we have a strong link to PAH levels present in Ms. Griffin, Derion's mother, and the development of this premature baby.

Q The mother is Jennifer Griffin; right?

A Yes.

Q Do you know how many other pre-term babies Jennifer Griffin had?

A Her other child Rahaeem was 7 months' gestation and born two months early and weighted 5 pounds and was not nearly as premature or as small as the brother.

Q Do you know how many full-term babies Jennifer Griffin has had?

A No. I don't have that information in front of me.

Q If Jennifer Griffin was heavily exposed to PAHs, would you expect all her babies to be born prematurely?

A It depends on the dose she sustained during the pregnancy and other factors, such as nutrition and other factors, so she could have a normal term baby.

Q Are there other factors besides PAHs which would contribute to Ms. Griffin for not carrying babies to full term?

A Well, she certainly was not old and she was 23 when she had Derion and age is not a factor. She's not a drinker -- well, she may be -- I don't have her history here. I have Derion's history. So I would have to say that there may be some contribution from other factors but I have not got them identified here at this point.

Q Is narcotics abuse a risk factor for delivering pre-term babies?

A By narcotics I guess you mean heroin?

Q More cocaine or amphetamines.

A Cocaine is a risk factor for having premature babies. Amphetamines, I don't know if it is or not.

Q What other risk factors are there generally for pre-term babies?

A Smoking cigarettes, heavy drinking, alcohol, probably drugs, if abused, probably would be on that list, cocaine being one associated with problems of early delivery and small babies. I'm not recalling off hand other risk factors and I'm sure there are others. Older mothers, and a family history of reproductive problems, some kind of genetic factors that play a role. That's all I can think of at the moment.

Q Are even some young women genetically predisposed to deliver early?

A Well, with the family history of say the mothers, sisters, aunts, grandparents and the mother having a history, it could be suggestive of a genetic effect and it can effect a younger woman.

Q Ms. Griffin's medical records indicate that she had a very early pregnancy of her first, which was terminated either spontaneously or otherwise?

A Correct.

Q Spontaneous or an intentional abortion and we're not sure which; is that right?

A As I understand it, it was probably an induced abortion but it might have been spontaneous. I'm not certain.

Q In any event, having an abortion at a very young age -- I believe she was in her early teens --

would that predispose someone to have pre-term babies later?

A No.

Q Is there any epidemiological literature that you're aware of on the effect of an abortion at a young age on later pregnancies?

A I think there is a study there that suggests that there is an increased risk but I don't think it's been borne out. I think there are many other studies that suggest that is not true.

Q Let's assume that Ms. Griffin did not have any PAH or dioxin exposure and yet delivered a baby at 27 weeks --

A 22 to 24.

Q 22 to 24 weeks, what health problems would you expect a baby who is born at that early a gestational age to have, irrespective of PAH and dioxin exposure?

A Just the prematurity alone increases their risk for developing developmental problems, achieving at school and developing the brain to its full maximal capacity, and they're more prone to various respiratory problems.

Q So intellectual achievement and respiratory problems?

A Those are the two that come to mind as a

consequence of severe prematurity in and of itself.

Q Any others?

A No.

Q Now, we've described Derion many time as hydrocephalic; correct?

A Correct. That's a diagnosis made early on in his life.

Q In layman's terms, what does that mean?

A It means there is a blockage to the drainage of the fluid in the brain, and they have to put a shunt in to relieve that pressure to create an artificial relief valve so that the fluid in the brain doesn't build up and create the condition known as hydrocephalous, which is water on the brain, and if that blockage occurs and is not treated, the head becomes very large and squeezes the brain and becomes severely retarded.

Q It's the actual physical squeezing of the brain down towards the opening of the base of the skull which causes the retardation; is that right?

A Well, the pressure of the whole brain prevents the growing and developing process.

Q It's a physical insult to the brain?

A It's a physical problem and it's solved by putting the shunt in.

Q Now, we began by talking about Derion Griffin's

health problems being related to PAH exposure, and I want to make sure we've completed that.

We talked about neurological problems and respiratory problems. Are there any other problems that Derion Griffin has that you believe are related to PAHs, dioxins or penta?

A I think I've indicated that he's got a picture similar to the other people living in the neighborhood, respiratory, skin, neurologic problems, and in addition to the extreme prematurity, he's been insulted by additional exposures during early life in living in this environment.

Q On page 9 of 9 of your summary of Derion Griffin, you identify dermal problems?

A Yes, as reported by his mother.

Q Are those related to the Koppers plant?

A I believe so. I didn't identify any other reason for the skin problems.

Q Do you think he'd have skin problems but for exposure to the Koppers plant or the PAHs, dioxins and penta?

A There is no reason to think otherwise and there is no other exposure to skin irritation, skin altering materials, and he has sort of a nonspecific skin complaint similar to what we've seen in our studies in

Columbus and the patterns that we see here in Grenada.
Q The next item this is on page 9 after skin problems is that chemicals from Koppers are known to cause cancer, lung disease, gastrointestinal disease, liver disfunction, autoimmune diseases, kidney problems, reproductive disorders, miscarriages, birth defects, premature birth, low birth weight and many other diseases and poor health.
Other than the low birth weight and premature birth, which of these problems does Derion have now?
A I think we're talking about the neurologic problems I alluded to, but I don't think that was on the list you just read, but I'd say in the next paragraph I talk about his neurological problems.
Q So Derion doesn't have cancer currently?
A Correct.
Q Or lung disease or gastrointestinal disease?
A Well, he has wheezing, shortness of breath, dry cough, productive cough, so I think he does have chronic bronchitis and probably a touch of asthma.
Q Does he have gastrointestinal disease?
A I didn't identify any GI complaints.
Q Does he have liver dysfunction?
A Not that I identified.
Q Does he have kidney problems?

A Not that I identified.
Q He doesn't have a reproduction disorder, does he?
A No.
Q Obviously, he's not prone to miscarriage; correct?
A Correct.
Q In the neurological complaints, you've indicated blurred vision, eye irritation, headache, loss of balance, irritability, lack of concentration, decreased short-term memory, decreased long-term memory, lack of coordination and slurred speech; correct?
A Yes.
Q Those are all reported by his mother?
A Yes. And also they were observed. When he tried to stand with his feet together, he tended to fall. He was very poorly coordinated and that's something I observed in my physical exam.
Q How do you know he has blurred vision?
A That was reported by his mother.
Q Same with eye irritation?
A Yes.
Q Headaches were reported by his mother?
A Yes.
Q Irritability was reported by the mother?

A Yes.
Q Lack of concentration, that was reported by the mother?
A Yes.
Q Decreased short-term memory was reported by the mother?
A Yes.
Q Decreased long-term memory was reported by the mother?
A Yes.
Q Slurred speech, did you actually hear him speak?
A He never really spoke. When I was talking to him, he would barely respond to commands. When I asked him to sit on the table or asked him to keep his eyes open, he wouldn't even do that. He's not very communicative, so I did not observe any speech at all.
Q Next is respiratory problems, and you have a list of respiratory problems, and those you believe were caused by his exposure to PAHs or dioxins?
A Yes. There is no doubt that prematurity, as I stated, increases the risk of having respiratory conditions later, but the interaction between the environmental pollution with his premature lung and respiratory system, I think the combination is why he has

these continuing problems.
Q But for exposure to PAHs, penta and dioxins, do you think Derion Griffin would have the same respiratory symptoms?
A I don't think he'd have the same respiratory symptoms, as a result of his prematurity, which I think is a result of his exposures. In a way it's kind of going in a circle.
But you're saying in the absence of his exposure post-delivery, after coming home from the hospital?
Q Right. Would he have these problems?
A I don't know. It's difficult to say whether prematurity alone would have caused any of his current problems. It's possible.
Q But children who are premature and don't live near the Koppers plant also have asthma and respiratory problems?
A Yes, they do.
Q Is childhood asthma something of an epidemic in this country?
A Yes.
Q Why is that, do you know?
A I think most scientists who are studying it believe it's due to the environmental pollutants that we

have in our environment. Studies done over the last five years by Dr. Peters at U.S.C. clearly demonstrate that the increased rate of asthma in the Los Angeles basin is related to the oxidant load present in their neighborhood.

Q So is it possible for you to opine that Derion Griffin would have had the same respiratory problems that he does if he had not lived in the neighborhood he does?

A Well, if he lived in east L.A., one of the polluted neighborhoods, he might have. But even then the rate is 12, 15 percent in high-risk neighborhoods.

But he lived in Grenada, Mississippi. If he lived in a non-polluted area, he probably would not have a high oxidant load because Grenada does not have a lot of smog buildup of pollutants like we do here in Los Angeles.

So, I mean, each person has their own unique situation. But the epidemic of asthma you talk about is more of a problem where there is actually some identifiable causes. It's not happening by magic and there is a reason. Same with Derion. He and his neighbors are so sick and it's because of a reason.

Q I have a nephew that lives in Hinckley, Illinois, which is in the middle of corn fields and soybean fields and he has childhood asthma.

Child get asthma irrespective of exposures; correct?

A Well, you just described a fairly polluted environment. I would not say living in the middle of a cornfield was free of all chemical agents by any means. My cousin farms a few thousands acres of corn in the middle of Nebraska and they use a lot of chemicals on those cornfields.

Q Are those the sort of children that get childhood asthma, children in agricultural areas?

A It certainly happens, yes.

Q And children that live in cities --

A They get it for other reasons.

Q How about the suburbs?

A Not everybody has asthma, and there is some interaction there. Cause and effect.

Q In the time you have left -- you want to leave at 4:00 o'clock?

A Right.

Q I want to switch to another subject and try to move into something else.

In your report you talk about dioxin exposures. You did blood tests on 29 people for dioxin?

A That's correct.

Q And then you report in, I believe, table 5 the

exposed mean dioxin and the unexposed mean dioxin for several individual congeners; is that correct?

A Yes.

Q Where did the unexposed mean dioxin levels you report on table 5 come from?

A I believe these were Greenville, Mississippi, if I remember correctly. I'm pretty sure that's what it is.

Q On another point in your report, and I know you're looking for it, and this is page 83, you mentioned controls Dallas equals 200.

A That's one set of controls, and I'm trying to remember which control values I used here. Page 83 -- that's pentachlorophenol. We didn't do pentachlorophenol in the Greenville people. We did dioxins and furans on them. This Dallas control value is for PCP, pentachlorophenol. This is Greenville, Mississippi where we did -- I think we did a total of 30 people.

Q Have you produced the results for the unexposed controls, that is the lab reports that show what the various detections were --

A I think we redacted the names but I think we gave you the data from -- this was done at AXYS Laboratories.

Q That's the AXYS lab report?

A Yes.

Q Are you familiar with a scientist named Arnold Schecter?

A Yes.

Q He has done some work on identifying levels of dioxin in people's blood; is that right?

A Yes.

Q And, in fact, you have relied on Dr. Schecter's work?

A He's one of the co-authors of our Columbus paper.

Q Let me mark this deposition Exhibit 2.

(Defendants' Exhibit 1 and 2 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Let me show you what's marked deposition Exhibit 2, a paper authored by Dr. Schecter.

Have you seen deposition Exhibit 2 before?

A Have I seen this paper before?

Q Yes.

A This is a poster he presented at -- I think the dioxin meetings back in probably '99 or something like that. The values here are from -- well, let's see. The German values were '89 and '94 and the U.S. values were up through '96, it looks like, blood samples.

Q Are you familiar with what Dr. Schecter did to obtain his U.S. values, where he went and got the blood and how many people contributed to the blood --
A Does he say here?
Q He does but I was wondering if you know the story.
A Well, he collected blood samples from patients all over the place. I don't remember where he did this one. He writes that "We collected and pooled samples from a young population, N=100, and an older population, N=100. These were from left-over samples at a Texas hospital which were collected for this study anonymously and which were ready to be discarded after the medical testing had been performed."
This is the 200 patients that we used as a comparison group for our study in Columbus.
Q You've seen these numbers before and know about the set of 200 people who contributed to the pooled blood samples that Dr. Schecter used?
A Yes, I did.
Q Was Dr. Schecter's purpose, to the extent you understand it, to obtain a sort of control value for various congeners of dioxins in people's blood in the United States?
A Yes, that's the purpose.

Q Was the idea, with the Dallas control population, that they were unexposed or were unknown exposure levels?
A I think unknown exposure. We know they were coming to the hospital and they were sick or most likely were sick with something, otherwise they would not have been in the hospital having blood drawn, and they were what I call a sick population.
Q And you used these numbers -- Dr. Schecter's numbers from his 2000 paper in studies you've done and papers you've written on dioxin exposure; is that right?
A Yes.
Q You've reported this Dallas cohort as your control population?
A Yes.
Q Do you have any sort of special access to Dr. Schecter's raw data?
A No. He just supplied me with the numbers, the pooled sample numbers.
Q You rely on Dr. Schecter's published numbers; is that right?
A Well, I don't -- he sent me the table with the numbers and I didn't go check and make sure they're the same as the published paper.
Q You don't know whether Dr. Schecter's

unpublished values are the same as what we see in the published paper?
A Well, I can probably dig around and find out. If you're talking about the poster that he made --
Q Exhibit 2.
A Whether those are the same as he gave me before?
Q Right.
A I don't know.
Q Is there any reason he would give you one set of numbers and published a different set?
A I don't think he would have, but I haven't checked.
Q Let's mark this as deposition Exhibit 3.
(Defendants' Exhibit 3 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Do you recognize Exhibit 3?
A Yes.
Q What is deposition Exhibit 3?
A Well, it's the paper we presented I believe in 2004.
Q You and Dr. Schecter and Olaf Paepke, among others, authored this paper?
A That's correct.

Q And does this paper relate to your study of health effects or at least dioxins and DNA adduct levels for the Grenada, Mississippi cohort?
A Yes.
Q In 2004 when you presented this paper, you had a list of exposed residents and we see No. 29 and that's the number you got from Grenada; right?
A Yes.
Q And the report has mean concentration range?
A Yes.
Q Then we have the Controls-Dallas, N=200?
A Yes.
Q Those are Dr. Schecter controls?
A Yes.
Q Let's look at deposition Exhibit 1, table 5 from your report.
Why are the control values different?
A I said they're from Greenville, Mississippi.
Q So you published a paper with Dallas controls and then in your expert report, in this case, you used Greenville, Mississippi controls; correct?
A That's correct.
Q Why?
A Well, because as Dr. Schecter and I reported this month in the Journal of Occupational and

Environmental Medicine, the results in 2005 were significantly lower in general population than in 1998, and that 6-year difference means that you've got to redraw and remeasure.

These Greenville, Mississippi patients were done contemporaneously or closer in time, in 2004, the same year we did the controls.

Number two, they were done in a Mississippi town similar in size and other factors to Greenville to make a better control.

Number three, we had questionnaire data on these people and we knew they didn't have any reason to have high values and didn't work in a place or live in a place where they have high dioxins so they'd be a better control.

The values are lower. The TCDD is not detectable. Many of the values are lower, as we just reported in the JOEM, reflecting the fact that there is a decline in the general population of dioxin levels.

Q In the JOEM, when you reported the general decline in the dioxin levels, would you say that in the last five years the general dioxin levels in the U.S. have declined by half?

A It's down by something in that order of magnitude or greater. It depends how far back you go. I

think we just reported they're down 83 percent from what they were 20, 25 years ago.

Q Now, Greenville, Mississippi, aren't you involved in litigation in Greenville?

A Yes.

Q The Platte chemical case?

A Yes.

Q How were the Greenville, Mississippi controls selected?

A Originally, one of the other experts in the case wanted to do dioxin levels in Greenville because he suspected there might be dioxins present, and we picked I think about 20 people who lived close to the Platte Chemical plant.

Then we had them identify a friend or relative who lived somewhere in Greenville, but far away from the plant who was otherwise the same age, sex, social economic level, and we matched them with the 20 people.

Then we took those two groups and divided them into ten and pooled ten samples, and ran two samples of our non-exposed Platte Chemical and a group of 20 pooled groups of ten who lived close to Platte Chemical.

The results in all four groups were identical and gave these values, and so we assumed there was no dioxin exposure going on in this group and, if you look

at the Dallas values and the decline over time, it fits nicely. It seems like very logical – the decline is not great but continuing.

Q So the Platte Chemical plaintiffs chose their own controls?

A We identified people who matched them and gave them questionnaire and made sure we knew everything about them. We asked them to identify someone similar to themselves but did not live in that neighborhood.

Q Have you ever done that in another case?

A It's a common way of doing controls.

Q Have you ever done it in any other case?

A No. We did it at the suggestion of our epidemiologist.

Q Who is that?

A Anderson.

Q What's the first name?

A Pamela Anderson.

MR. HOPP: We can mark this as 4.

(Defendants' Exhibit 4 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Do you recognize deposition Exhibit 4?

A Yes.

Q What's deposition Exhibit 4?

A That's our exposure assessment paper for Columbus, Mississippi.

Q In your exposure assessment for Columbus, Mississippi, you also report control values for TCDDs?

A Yes.

Q And various other congeners of dioxin; correct? Look at table 2, page 104.

A Right.

Q And you rely on Dr. Schecter's Dallas cohort; is that correct?

A Yes. That's what it says here. We utilized the Dallas controls as one of the comparisons we made.

Q And Dr. Schecter's paper, which we marked as deposition Exhibit 2, was published prior to your exposure assessment published in 2003; is that correct?

A Correct.

Q Let's flip to Dr. Schecter's paper, deposition Exhibit 2, and leave Exhibit 4 open to page 104.

A All right. You want to look at table 5 in this one?

Q Table 2 in both documents. Now, in Dr. Schecter's paper, the 2000 paper, he reports two different values for the Dallas cohort?

A Yes.

Q And looking at specifically TCDD, he reports

4.3 and 4.2.
Do you see that?
A Yes.
Q And your paper from 2003, you report on table 2, General Population Dallas; Schecter 2000, N=200, a value of TCDDs of 2.6?
A Yes.
Q How did you go from Dr. Schecter's values to your values?
A I don't know. I don't see how you can combine those to get to 200 and have a value to be lower, and I have to ask Dr. Schecter why the discrepancy.
I do have an explanation, which I think may be that the 200 people we got were different than the 200 people in this group, and that's one possibility.
Q This is the second Dallas control group of 200 people?
A Yes. In this case he had a hundred young people and a hundred older people, and in this group he didn't make that distinction of age and lumped them all together, I suspect. I'd have to check with him but I suspect it's two different groups he's pooled.
He has a deal with the lab there at UT Southwestern and he can get discarded blood fairly easily, so when he pools it, he sends it to Dr. Paepke

in Germany and he runs it for him. And just looking at these numbers that are consistently lower, I would think they were probably drawn at a later time.
Q So you think --
A That's -- I have to ask him.
Q So you think at some other point in 2000, Dr. Schecter developed another cohort of 200 people that he had evaluated?
A Yes. In other words, the Schecter references that I give here -- I have to ask him about that. I don't know how to explain the discrepancy at this moment.
Q What's the Schecter reference that you give in your 2000 paper?
A There are several Schecter references but none of them are 2000.
Q So you don't cite Dr. Schecter's 2000 paper in your 2003 paper in environmental research?
A No. This was personal communication. I'm guessing now that these are values he collected in 2000, and others were collected in -- it looks like 1996, four years before.
Q Somewhere in your office, in your possession, do you have a file of materials that provided the backup for your 2003 paper?
A Yes.

MR. HOPP: And we'll send a request, but I'd like to make a request on the record that we at least get that bit of it. I'd like Dr. Schecter's set of numbers. I'd like to see those.
Let's mark this Exhibit 5.
(Defendants' Exhibit 5 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Deposition Exhibit 5, can you tell me what it is?
A Let's see when did we do this one. I think this is a paper we presented at the dioxin meeting 2001. I'm not sure about that, but I believe we did. It's the same values we had in the paper.
Q Let's look at that. There is no table number. The table in deposition Exhibit 5 which list dioxin congeners --
A Yes.
Q Again, you have control values, General Population Dallas; Schecter 2000, and you have numbers different from the numbers in the Schecter 2000 paper.
Do you see that?
A Yes, these are the same ones that we had in our paper we were just looking at.
Q They're the same for the 2003 paper, except for

one. Let's look at control value for 1,2,3,7,8 PeCDD.
Do you see that?
A Yes. 6.3?
Q I'm looking at the control value in the 2000 paper -- in the 2003 paper, deposition Exhibit 4, the control value is point 05?
A For the penta?
Q 2,3,7,8 penta.
A Yes. 6.3 for the control value -- same on both.
Q We're looking at two different numbers. Let me --
A Just point it out --
Q This number here. Do you see that?
A All right.
Q It's see point 05 on the 2003 paper, and in your paper that we have designated Exhibit 5 it is point 5, so we're off by an order of magnitude; correct?
A That's right. But it's probably a typo because all the other numbers, including the TEQs, are the same and it's just a typo.
Q Which document is correct?
A Oh, I suspect it's the point 5 rather than point 05. The detection limits are such that it's not likely to be point 05. I think point 5 is the correct

one. The TEQ value or the TEF value really is point 05 and someone just transposed the number.
Q You think someone made a transcription error?
A I think so. If you go down to the total TEQs and the other totals, they're the same. Yes -- if it was an error, it was carried through -- it's just a typo.
Q If would change the TEQ?
A Yes, if it was anything other than that.
(Defendants' Exhibit 6 was marked for identification by the court reporter.)
BY MR. HOPP:
Q You have deposition 6 in front of you?
A Yes.
Q What is that?
A A presentation that I made in Berlin in September of 2004.
Q Look at table 1, page 2844.
A Okay.
Q You have your exposed population control and then a control equals 100 population.
Do you see that?
A Yes.
Q Where did the control values come from in the paper we have as Exhibit 6?
A Well, this was yet a different set published by

Paepke. He gave us, along with the reports in this case, he gave us this. This is his reference population for that set of bloods. So we didn't use the same control values. They're similar and a little higher on the TCDD but similar, in general, to what we've had here.
Q So the citation -- and correct me if I'm wrong -- the citation for the control values in deposition Exhibit 6 -- I believe it's footnote 5 -- which is Schecter, A.J.Paepke O. And Piskac A.L. (2000) Organohalogen Compounds 48,68-71.
Do you see that?
A Yes. That's the citation we were talking about earlier, isn't it?
Q Deposition Exhibit 2 --
A This is the older and definitely out of date values but again it doesn't quite match.
Q Doesn't match up at all, does it?
A No, it doesn't.
Q Why is that?
A Like I said, these values were from Paepke, and he gave us these values as a reference range. So I suspect, given how high they are, they're German values.
Q Do you have somewhere a file of backup materials for your paper on Persistent Organic Pollutants in the 9/11 World Trade Center rescue workers?

A Yes.
Q I'd like a copy of the backup for the control values for that paper, and I'll sent a written request.
Why in 2004 were you reporting 5-year-old control population data in your paper on the 9/11 World Trade Center rescue workers and reporting more recent data for the Greenville -- Grenada population?
A I got the Greenville data after we submitted this paper, so I didn't have the Greenville controls for the firemen, and we didn't have a good control for the firemen, anyway, and we used -- given the fact it references this document, but they don't match, and what I see here is values that are very similar to what is listed here -- they're in the same order of magnitude but not identical.
Q They're different?
A The TEQ's are quite high.
Q In the unexposed population?
A I know what it is. The PCBs are not done here. This is Paepke's normals and this reference should reflect Paepke's -- as I said, these are normals he sent along with the reports on these firemen, and that's -- well, I have to dig into this and figure out exactly what he did here, but I believe that the controls were not from this 2000 paper.

Q Once again, I'd like to see the control data that Dr. Paepke supplied to you.
A These pages do refer to this but the numbers don't match.
Q The citation is to Dr. Paepke's and Dr. Schecter's 2000 paper?
A Let's see. If we look over here to -- no, it still doesn't match. I'll get to the bottom of this.
Q In your table number 5 for the Grenada cohort, your mean control value for TCDD is non-detect?
A Yes.
Q For clarity of the record, that means in whatever lab report you got back on your controls there was no TCDD detected in anybody's blood?
A Yes.
Q Have you ever seen an absence of TCDDs in a sample of 29 people?
A Well, there is four pooled samples, and this is our unexposed controls in Greenville, and it depends what your detection limits are and they're getting -- as I said, PCB levels are dropping significantly in the United States and getting close to detection level and, even though I've not seen it before, I believe it's position.
Q What was the detection limit you used for your control population that you see reflected on --

A I have to get out the reports from the lab to answer that. I don't have that information.
Q We'll look at that information tomorrow and we're almost out of time for today. If you remember, it's something I'd like you to pull?
MR. HOPP: This is Exhibit 7.
(Defendants' Exhibit 7 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Do you recognize deposition Exhibit 7?
A Well, it looks like a draft or a manuscript of the paper we submitted. I don't know if it's the one published or an earlier version.
Q The document we see set forth is deposition Exhibit 7. Do you know if this was in effect your expert report in the Columbus, Mississippi litigation? Is this your expert report?
A No. This is a manuscript I think that we submitted to one of the journals of the paper.
Q Do you believe then that -- here we go. It says to send all page proofs to James Dahlgren, M.D. Do you see that?
A Yes.
Q That's a sort of language you have on a document you submit for publication?

A Yes.
Q Was Exhibit 7 ever rejected for publication?
A Well, we sent it to Occupational and Environmental Medicine and they rejected it, and then sent it to Environmental Research and they published it. So I don't remember from memory if this is what we sent to Occupational and Environmental Medicine or not.
Q You received reviewer's comments and a rejection from Environmental Research?
A No. From Occupational and Environmental Medicine, formerly the British Journal of Industrial Medicine.
Q And Exhibit 4 is the second paper, which was then submitted to Environmental Research?
A Right. Well, the paper that got published.
Q You added an author in deposition Exhibit 4 that we don't see on Exhibit 7, Harpreet Takhar?
A Yes.
Q Who is he?
A An epidemiologist who works with me.
Q Does he work for your company?
A Yes.
Q You employee him?
A Yes.
Q What was his role, if any, in the version we

see reflected in deposition Exhibit 4?
A Well, he helped us write the paperwork on it, and I don't remember specifically what he did but he helped us with it.
Q Were there a lot of changes made between the versions we see in deposition 7 and the version we see in deposition 4?
A No. I think there were some changes, but I don't know if I'd characterize them as many but there were some changes, and you can read the two papers and see they're substantially similar but probably there are some differences.
Q Is it standard practice to submit a paper to a journal where the scientist is also a peer reviewer?
A You mean peer review your own paper?
Q No. You're on the list of peer reviewers for a journal. Is it standard practice to submit papers to that journal?
A Sure. We do that all the time.
Q At the time your paper was published in Environmental Research, were you a peer reviewer for Environmental Research?
A No. He asked me to be a peer reviewer after that.
Q And who is the publisher of Environmental

Research? I mean, who is the editor?
A The editor is Ellen Silbergeld.
Q Where does he work?
A She works at Johns-Hopkins.
Q Ellen Silbergeld?
A Ellen Silbergeld.
MR. HOPP: It's 4:00 o'clock on the nose. Shall we break?
THE WITNESS: Yes.
MR. HOPP: I'm ordering this written and I'd like the original.
MR. LUNDY: A copy, yes.

PENALTY OF PERJURY

I hereby declare I am the deponent in the within matter; that I have read the foregoing proceeding and know the contents thereof and I declare that the same is true of my knowledge except as to the matters which are therein stated upon my information or belief, and as to those matters I believe it to be true.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on the _____ day of _____, 2005, at _____, California.

_____

JAMES DAHLGREN, M.D.

I, VIRGINIA PETERAITIS, a certified shorthand reporter for the State of California, do hereby certify:

That prior to being examined the witness named in the foregoing deposition, was by me duly sworn to testify the truth, the whole truth, and nothing but the truth pursuant to Section No. 2093 of the Code of civil Procedure;

That said deposition was taken before me pursuant to notice, at the time and place therein set forth, and was taken down by me in shorthand and thereafter reduced to typewriting via computer-aided transcription under my direction;

I further certify that I am neither counsel for, nor related to, any party to said action, nor in anywise interested in the outcome thereof.

IN WITNESS WHEREOF, I have hereunto subscribed my name this ___ day of _____, 2005.

_____

VIRGINIA PETERAITIS
CSR No. 6205